

CASE REPORT

Is ventricular ectopy a legitimate target for ablation?

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Abstract

Radiofrequency ablation has an established role in the treatment of non-ischaemic ventricular tachycardia. A few patients present with symptomatic but benign ventricular ectopy that can be mapped to the right ventricular outflow tract. The successful use of radiofrequency ablation in a patient with drug resistant, symptomatic ventricular ectopy is reported. Radiofrequency ablation may have a useful role in more benign arrhythmias.

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Radiofrequency ablation is now an established tool in the treatment of cardiac arrhythmias.^{1,2} Initially it was used solely in tachycardias associated with pre-excitation syndromes,^{3,4} but it is now used in a broad variety of atrial and ventricular arrhythmias. However, there has been little information on the use of ablation in patients with "benign" ventricular ectopy.⁵ We report the successful use of radiofrequency ablation in a patient with persistent and symptomatic ventricular ectopy and discuss the implications.

Case report

A 50 year old woman presented with a two year history of recurrent palpitation, atypical chest pain, dizziness, and breathlessness at rest and on exercise. Other than a long history of smoking, she had no other history of note and physical examination was unremarkable.

An electrocardiogram showed regular unifocal ventricular ectopy but was otherwise normal. A 24 hour ambulatory monitor showed frequent unifocal ectopy, runs of bigeminy, couplets, and brief three beat salvoes of ventricular tachycardia. A signal averaged electrocardiogram was normal. On exercise testing, she achieved maximal heart rate with no evidence of myocardial ischaemia. Ectopic activity was abolished by exercise. Echocardiography showed normal chamber dimensions, normal valves, and normal right and left ventricular function. Chest x ray, antibody titres, viral screens, routine biochemistry, and haematology were all normal. She was reassured of the benign nature of her arrhythmia and discharged on a

trial of sustained release mexiletine 360 mg twice a day.

Her symptoms persisted and subsequent trials of oral propafenone, flecainide, and verapamil over the next 18 months did not abolish ectopy or improve symptoms. Her persistent complaints of severe and debilitating symptoms led to her admission to hospital for further investigation.

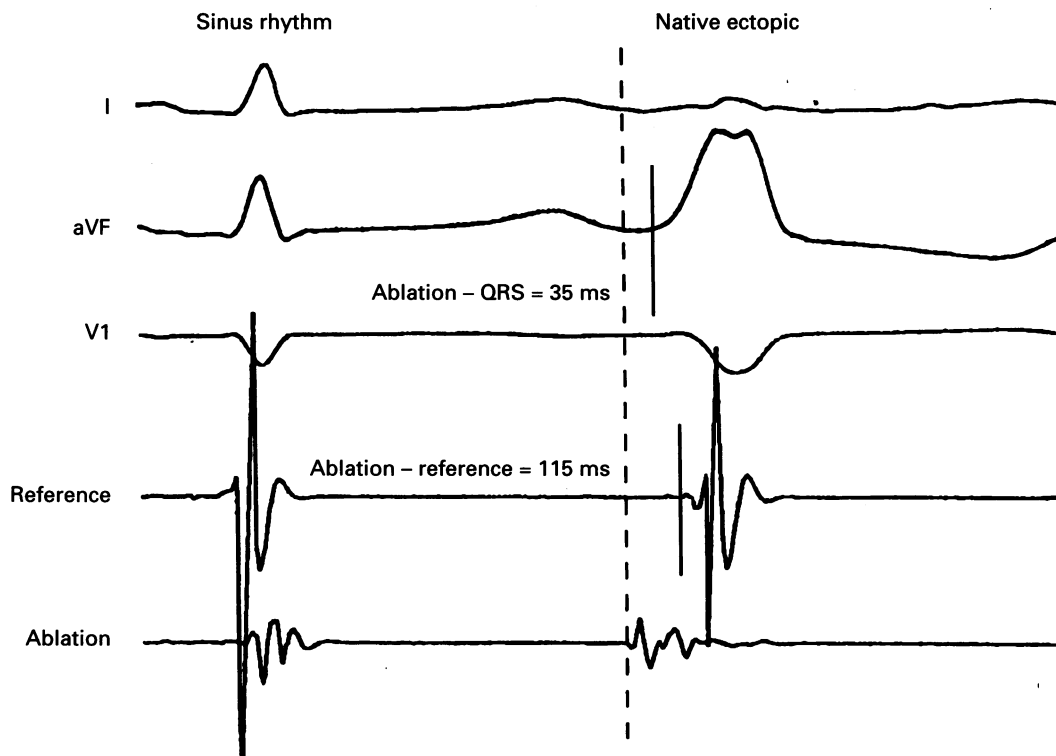
A 12 lead electrocardiogram again showed unifocal ventricular extrasystoles with a left bundle branch block configuration and a normal axis. Coronary angiography and left and right ventriculography performed to exclude unrecognised coronary artery disease were normal. Programmed electrical stimulation was performed with quadripolar catheters placed in the high right atrium (Polaris, Webster Laboratories) and the right ventricular apex (Josephson-Bard). Stimulation at 600 and 400 ms drive cycle lengths in both the right ventricular apex and outflow tract did not induce sustained arrhythmia. Isoprenaline infusion (up to 10 µg/min) induced an increased frequency of the ectopy but not sustained arrhythmia. The procedure was then extended to map the ventricular ectopy.

The onset of QRS complex in the surface electrocardiogram and the right ventricular endocardial signal were used as fixed point references and the ablation catheter was moved to the right ventricular cavity. We used an on-line digital system to time the ectopic signals from the roving ablation catheter in relation to the two references and to guide mapping of the extrasystoles. We used biplane screening in the 30° right anterior and 60° left anterior oblique positions. This technique showed that the extrasystoles were localised to the right ventricular outflow tract. At several sites throughout the tract, ectopic signals from the ablation catheter preceded the onset of the QRS complex and the right ventricular apical signal by up to 30 ms and 110 ms respectively. At a single and reliable site, posterior to and immediately below the pulmonary valve, signals maximally preceded the surface and apical signals by 35 ms and 115 ms respectively (fig 1). Pace mapping at the lowest possible voltage at this site provided an exact 12 lead match of the ectopic configuration (fig 2). After intravenous heparin (10 000 IU) radiofrequency energy (Radionics-3D) was

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Figure 1 Endocardial and surface lead signals at the site of successful ablation.



applied to this site (30 W for 45 s) under continuous screening, without impedance rise or displacement. When no extrasystoles were seen over the subsequent five minutes, a second "consolidation burn" (30 W for 60 s) was applied to the same site. After 20 min and no further extrasystoles, programmed stimulation and isoprenaline infusion protocols were repeated, during these no arrhythmia was seen. The total screening time was 30 min. The procedure was uncomplicated and the patient was discharged well 24 hours later. Ambulatory monitoring after discharge showed no arrhythmia and the patient was well and free from cardiac symptoms 4 months after the procedure.

Discussion

Many patients complaining of troublesome palpitation are found to have ventricular ectopy in the presence of a "normal" heart. There is often accompanying atypical chest pain, dizziness, or dyspnoea. The mechanism of these symptoms is unclear but may be related to undetected sustained arrhythmia or to functional tricuspid and mitral regurgitation during the arrhythmia. Conventional treatments based on reassurance or serial drug testing or both are often unsuccessful, usually as a result of drug failures or side effects.

Radiofrequency ablation has become a viable alternative to drug treatment in non-ischaemic ventricular tachycardia, successfully destroying the anatomical substrate—often an abnormal focus with enhanced automaticity in the right ventricular outflow tract.^{6,7} Whereas its role is readily justified in incessant or non-sustained tachycardias, it has not been widely used in ventricular

ectopy. Nevertheless, it seems reasonable to consider symptomatic patients with more benign automatic arrhythmias arising from the same area as candidates for ablation. Because exercise, isoprenaline, or programmed stimulation did not provoke the tachycardia the use of ablation would not have been warranted in this case according to conventional practice. Nevertheless, we believed that the monomorphic ectopy arose from an automatic focus similar to that responsible for a sustained automatic ventricular tachycardia and was therefore a legitimate "target" for ablation.

The techniques of ectopic mapping and ablation are similar to those used in ventricular tachycardia. Identification of the earliest site of activation during the extrasystole and an exact match during pace mapping are both used in identifying the generator focus. In this case, ectopic activity was easily traced to the right ventricular outflow tract by a roving catheter. During detailed mapping, on-line digital timing proved invaluable in identifying the focus where the onset of the ectopic QRS maximally preceded surface and endocardial QRS signals. The technique avoids the necessity for repeated measurements "on paper" and reduces screening times to a minimum.

As in many reported cases of sustained ventricular arrhythmia, the ectopic focus in this case was mapped to the right ventricular outflow tract. Why this site should be responsible for so many non-ischaemic arrhythmias is unknown, but may be related to the embryological origin of the tract. An endocardial location of such a focus is strongly suggested by the prompt abolition of arrhythmia early during radiofrequency application.

On the basis of this unique case, it would be inappropriate to suggest ablation is a first

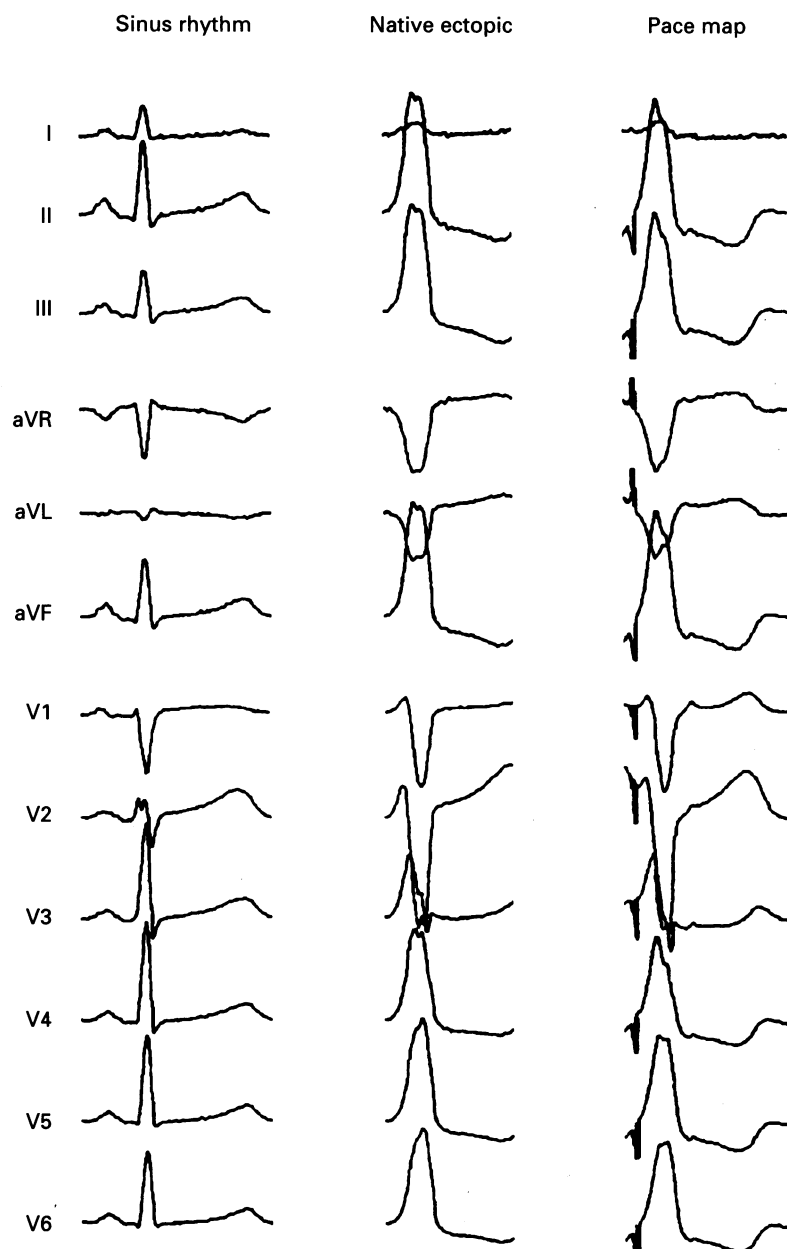


Figure 2 12 lead electrocardiograms of sinus rhythm, the "native" ectopic complex, and a pace map at the site of successful ablation.

line treatment for ventricular ectopy in patients with normal hearts or to imply that its use is appropriate in patients with organic heart disease. Our patient was carefully selected for radiofrequency ablation: she was disabled by symptoms clearly related to her arrhythmia and several attempts at drug treatments had failed. Furthermore, detailed electrophysiological mapping identified a discrete generator site in an area known to be associated with such arrhythmias. The indication for ectopic ablation remains limited to ectopy arising in the right ventricular outflow tract of patients with normal hearts in whom medical treatment has failed.

Conclusion

Many patients with otherwise normal hearts present with a range of ectopic ventricular activity arising from the right ventricular outflow tract. We suggest that this case exemplifies a very small subgroup of patients in whom automaticity is less marked and results in apparently benign arrhythmias and where ablation is the appropriate management.

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